

**REMARKS**

Reconsideration and continuing examination of the above-identified application is respectfully requested in view of the amendments above and the discussion that follows.

Independent claims 1, 2, 5, 33, and 63 have been amended to speed prosecution and further clarify the claimed subject matter. Claim 3 has been canceled. Claims 1, 2, 5-8, 33, 63, and 64 are pending.

**I. The Amendments**

Claim 3 has been canceled to speed prosecution.

Claims 1, 2, 5, 33, and 63 have been amended to speed prosecution by addition of the term 'purified' in reference to the TGF $\alpha$  polypeptide. Support for the addition of this term is found, at least, on page 51 wherein it is stated that the rat TGF $\alpha$  was obtained from Sigma Chemical Co. Enclosed Exhibit A contains copies of the cover, copyright page and page 1274 from the 1995 issue of the Sigma catalogue in which the rat TGF $\alpha$  is noted to be 90% pure. The independent claims have also been amended to improve their syntax by removing a redundant phrase.

It is thus seen that no new matter has been added.

**II. Rejection under 35 U.S.C. §102**

The Office Action dated December 12, 2003, rejected claims 1-3, 5-8, 20, 33, 63, and 64 under 35 U.S.C. §102(e) as being anticipated by Weiss et al. US Patent No. 5,980,885, filed June 7, 1995 and issued Nov. 9, 1999 (Weiss). As noted in the Examiner's Answer, claim 20 was cancelled in an amendment after final mailed on April 22, 2005. In light of the remaining claims, 1-3, 5-8, 33, 63, and 64 this rejection is again respectfully traversed.

**A. Administration of purified TGF- $\alpha$  polypeptide to a site outside of the ventricles**

In regard to the method of administration taught by Weiss, the Action notes that

Weiss et al., teach that the administration of the growth factors can be done by any method, including injection cannula, transfection of cells with growth hormones-expressing vectors, injection, and timed release apparatus which can administer substances at the desired site, see in particular column 25, lines 40-column 26, line 15. [page 4, Action, February 23, 2004]

The Action followed that statement with an excerpt from an earlier portion of Weiss, column 23, lines 4-21. "Such sites include transplantation to basal ganglia [sic], caudate, putamen, nucleus basalis or substantia nigra, i.e., into the striatum as claimed, see in particular column 23, lines 4-21". [page 4, Action, February 23, 2004.]

It is respectfully submitted that the excerpts from Weiss et al. have been isolated from their original disclosure and are taken out of context, whereas they should rather be taken in the context of their surrounding disclosure in its entirety. Taking in the entirety of the disclosure, in addressing the method of administration, the latter portion of Weiss cited in the Action (column 25, line 61 - column 26, line 15) is shown below:

The fact that neural stem cells are located in the tissues lining ventricles of mature brains offers several advantages for the modification and manipulation of these cells in vivo and the ultimate treatment of various neurological diseases, disorders, and injury that affect different regions of the CNS. Therapy for these can be tailored accordingly so that stem cells surrounding ventricles near the affected region would be manipulated or modified in vivo using the methods described herein. The ventricular system is found in nearly all brain regions and thus allows easier access to the affected areas. If one wants to modify the stem cells in vivo by exposing them to a composition comprising a growth factor or a viral vector, it is relatively easy to implant a device that administers the composition to the ventricle and thus, to the neural stem cells. For example, a cannula attached to an osmotic pump may be used to deliver the composition. Alternatively, the composition may be injected directly into the ventricles. The neural stem cell progeny can migrate into regions that have been damaged as a result of injury or disease. Furthermore, the close proximity of the ventricles to many brain regions would allow for the diffusion of a secreted neurological agent by the stem cells or their progeny. [Emphasis supplied]

Therefore, it is seen that the intentions as to the location of the injection cannula, transfection of cells with growth hormone-expressing vectors, injection and timed-release apparatus taught by Weiss are solely directed to the ventricles.

Furthermore, the Action cited Weiss as teaching administering via oral administration (column 25, lines 40-55). Oral administration is a method clearly different from a method directed to parenteral administration of a TGF $\alpha$  polypeptide as is recited in the claims. The recitation of "parenteral" in the 27<sup>th</sup> Edition of Dorland's Illustrated Medical Dictionary (1988) page 1231 attached as Exhibit B is defined as 'not through the

alimentary canal but rather by injection through some other route, as subcutaneous, intramuscular, intraorbital, intracapsular, intraspinal, intrasternal, intravenous, etc'. Therefore, it is respectfully submitted that the teaching of oral administration as a prior disclosure of the presently and previously claimed parenteral administration is in error.

Furthermore, the undersigned is unaware of any FDA approved medicinal proteinaceous material that is to be taken orally for treatment of other than the gastrointestinal (GI) tract, let alone an orally administered peptide that is said to successfully pass through the GI tract and into the CNS, to illicit a therapeutically effective response. It is submitted that human bodies are designed to prevent successful administration of such proteinaceous materials. It is further submitted that without a more complete description of how to accomplish successful oral administration of a peptide claimed here, Weiss does not enable oral administration. Should the Examiner maintain this basis for rejection, it is requested that documentary evidence be provided that illustrates the successful and approved oral administration of a proteinaceous material to an individual.

In addition to the method of administration, the Action addressed the site of administration, the Action cited column 23, lines 4-14 shown below:

Transplantation can be done bilaterally, or, in the case of a patient suffering from Parkinson's Disease, contralateral to the most affected side. Surgery is performed in a manner in which particular brain regions may be located, such as in relation to skull sutures, particularly with a stereotaxic guide. Cells are delivered throughout any affected neural area, in particular to the basal ganglia, and preferably to the caudate and putamen, the nucleus basalis or the substantia nigra. Cells are administered to the particular region using any method which maintains the integrity of surrounding areas of the brain, preferably by injection cannula. [Emphasis supplied.]

It is thus seen that the administration sites listed in the Action, when read in more complete context, are in reference to transplantation of cells generated from dissociated tissues and proliferated *in vitro*. See for example:

[t]he method comprises the steps of administering growth factors to a mammal to induce *in vivo* proliferation of neural precursor cells, removing the precursor cell progeny from the mammal, culturing the removed cells in vitro in the presence of one or more growth factors that induces multipotent neural stem cell proliferation, and implanting the multipotent neural stem cell progeny into the mammal. (Abstract, 1st sentence:Emphasis supplied.)

Transplantation of cells significantly differs from the manipulations of the pending claims that are directed to administration of purified TGF $\alpha$  polypeptide or functional fragment thereof.

It is respectfully submitted that it is inappropriate to isolate passages from Weiss et al. and rearrange them to create a disclosure that appears to mimic the currently amended claims. Weiss et al. teaches administration of cells

proliferated *in vitro* via grafting and transplantation to various regions of the brain. Weiss also teaches administration of growth factors or vectors via injection in the ventricles. The current amended claims are directed to parenteral administration of purified TGF $\alpha$  polypeptide or a functional fragment thereof, and therefore to a site outside the ventricles, a method clearly different from that disclosed by Weiss.

**B. Weiss is not a proper reference for TGF $\alpha$   
due to a lack of an enabling disclosure**

Although under MPEP 2121, the disclosure of a prior art reference is presumed to be enabling, it is submitted that under MPEP 2121.01, "[t]he disclosure in an assertedly anticipating reference must provide an enabling disclosure of the desired subject matter; mere naming or description of the subject matter is insufficient, if it cannot be produced without undue experimentation."

No specific instructions as to the use of purified TGF $\alpha$  to induce migration are provided by Weiss. A list of proliferation-inducing growth factors is provided that includes TGF $\alpha$  [Weiss, column 16, line 6].

It is submitted, however, that reciting transforming growth factor (TGF $\alpha$ ) in a laundry list of exogenous growth factors that can be used to influence the differentiation of precursor cells *in vitro* is not enabling of the present *in vivo* invention. No specific use of TGF $\alpha$  *in vivo* to induce migration of neural stem cells or progeny thereof upon administration of purified TGF $\alpha$  to a site outside the ventricles is addressed in Weiss' disclosure and/or examples. MPEP 2121.01 states:

[a] reference contains an "enabling disclosure" if the public was in possession of the claimed invention before the date of invention. "Such possession is effected if one of ordinary skill in the art could have combined the publication's description of the invention with his [or her] own knowledge to make the claimed invention." *In re Donohue*, 766 F.2d 531, 226 USPQ 619 (Fed. Cir. 1985).

Weiss provides 45 examples in the specification. None of those 45 examples instructs or suggests to one skilled in the art how to use purified TGF $\alpha$  for *in vivo* applications. No examples suggest the use of purified TGF $\alpha$  for induction of migration to a site of CNS damage or injury via administration outside the ventricles. No instruction is provided by Weiss for administration of TGF $\alpha$  to a subject. Therefore, it is submitted that there is no demonstration that the public was in possession of the claimed invention prior to Applicants' filing date and that, under MPEP 2121.01, Weiss is not an effective, enabling reference. Therefore, the rejection 35 U.S.C. §102 should be withdrawn.

**C. Weiss is a contradictory teaching as to the  
use of growth factors and is thus not enabling**

Weiss discloses the use of growth factors in multiple portions of the specification. In various portions, Weiss contradicts its' own teachings and is thus not enabling to one of ordinary skill in the art.

For example, at column 25, lines 48-55, Weiss states:

[t]he neural stem cells can be induced to proliferate and differentiate *in vivo* by induction with particular growth factors or pharmaceutical compositions which will induce their proliferation and differentiation.

Therefore, this latter method circumvents the problems associated with transplantation and immune reactions to foreign cells. Any growth factor can be used, particularly EGF, TGF.alpha., FGF-1, FGF-2 and NGF. [Emphasis supplied.]

With respect to example 43, Weiss addresses the use of growth factors not only to proliferate and differentiate *in vivo*, but also for their use as regulatory factors and discusses their neurosphere-inducing properties. A list of regulatory factors used in Example 43 is shown below:

[a]ctivin, BMP-2, TGF- $\beta$ ., IL-2, IL-6, IL-8, MIP-1 $\delta$ , MIP-1 $\beta$ , MIP-2 (all obtained from Chiron Corp.), TNF- $\alpha$ , NGF (Sigma), PDGF (R&D Systems), EGF and CNTF (R. Dunn and P. Richardson, McGill University) were made up in separate flasks of compete [sic] medium to a final concentration of 0.2  $\mu$ g/ml. Retinoic acid (Sigma) was added at a concentration of 10<sup>-6</sup> M. 10  $\mu$ l of one of these regulator factor-containing solutions was added to each proliferative factor-containing well of the 96 well plates. Control wells, containing only proliferative factors, were also prepared.

In another set of experiments, the neurosphere inducing properties of each of these regulatory factors was tested by growing cell in their presence, in proliferative factor-free Complete Medium. None of these regulatory factors, with the exception of EGF, when used in the absence of a proliferation-inducing factor such as EGF or FGF, has an effect on neural stem cell proliferation. [Weiss, column 55, lines 21-64; Emphasis supplied.]

In comparing the two underlined portions of the separate quotations, above, it is apparent that the teachings of Weiss in regard to the use of growth factors are contradictory in the function of growth factors. This is specifically in regard to the use of NGF, a growth factor that is taught by Weiss to be a proliferative-inducing factor and a regulatory



factor. In the first provided quote, Weiss states that any growth factor, "particularly EGF, TGF.alpha, FGF-1, FGF-2 and NGF" [emphasis supplied], may be used to induce proliferation. In the second provided quote, Weiss states that when testing the use of regulatory factors, "[a]ctivin, BMP-2, TGF- $\beta$ , IL-2, IL-6, IL-8, MIP-1 $\delta$ , MIP-1 $\beta$ , MIP-2 [...], TNF- $\alpha$ , NGF [...], PDGF [...], EGF and CNTF" [emphasis supplied] as regulatory factors, that "none of these regulatory factors, with the exception of EGF, when used in the absence of a proliferation-inducing factor such as EGF or FGF, has an effect on neural stem cell proliferation".

It is therefore submitted that in focusing on the activity of NGF in the context of the provided quotes, Weiss addresses the use of growth factors in a contradictory manner, stating that NGF can be used to induce proliferation in the specification and later stating the NGF has no effect on neural stem cell proliferation. It is respectfully submitted that providing those skilled in the art with a contradiction does not place them in possession of the invention. The Weiss disclosure therefore does not meet the enablement requirements of MPEP 2121 in regard to the present claims, and cannot serve as a prior art reference against these claims. Accordingly, the rejection under 35 U.S.C. §102 should again be withdrawn.

### **III. Rejection under 35 U.S.C. §103:**

#### **Non-Obviousness Proven by Others of Ordinary Skill in the Art's Citations to the Present Invention**

The non-final Office Action dated December 12, 2003, rejected claims 1-3, 5-8, 33, 63, and 64 under 35 U.S.C. §103(a) as being obvious from Weiss et al. US Patent No. 5,980,885, filed June 7, 1995 and issued Nov. 9, 1999 (Weiss). This rejection is again respectfully traversed.

The previously provided arguments of the prior responses and Brief on Appeal for patentability are hereby reiterated by reference. In addition, having shown that the present claims are not anticipated, the present response is provided to improve the clarity of the claims as well as to provide further insight into the importance of the present invention to the scientific community and thereby further illustrate the novel and non-obvious status of the subject matter claimed herein.

**A. Citations by others in peer-reviewed literature to Fallon et al. PNAS 2000 establish scientific contribution and non-obviousness of the presently claimed invention**

In one of its first cases, the Court in *Stratoflex, Inc. v. Aeroquip Corp.*, 713 F.2d 1530, 1538, 218 USPQ 871, 879 (Fed. Cir. 1983), noted "[i]t is jurisprudentially inappropriate to disregard any relevant evidence on any issue in any case, patent cases included. Thus evidence arising out of the so-called 'secondary considerations' must always when present be considered en route to a determination of obviousness." In a situation such as that here where there have been no commercial activities 'secondary considerations' can be difficult to find. However, one measure of the impact of one's work upon the scientific community is the number of times and the manner in which that that work is cited by one's peers in the scientific community; i.e., those active and skilled in the pertinent art as they report upon work that they themselves have done. This assessment is borne out in the below-quoted portion from page 54 of the book by William Broad and Nicholas Wade, Betrayers of the Truth, Simon and Schuster, New York (1982) (attached as Exhibit C). Those authors state:

Jonathan and Stephen Cole, sociologists who in 1972 published a trenchant analysis of scientific productivity entitled "The Ortega Hypothesis,"<sup>11</sup> have concluded that only a few scientists contribute to scientific progress. The majority publish work that has little or even zero impact on the forward march of knowledge. The Coles' survey was based on the fact that a scientist is obliged to acknowledge in his published work at all the articles that have contributed to his own. The footnotes of references to other work, known as "citations," offer a powerful means of telling who influenced whom. [Emphasis supplied. A copy of the article of footnote 11, above, is attached hereto as Exhibit D.]

The data and concepts of the present application are in large part present in an article published after the present filing date of this application. The publication occurred in one of the world's most important scientific journals, the *Proceedings of the National Academy of Sciences, USA* (Fallon et al. PNAS 2000).

Although it is believed that the Fallon et al. PNAS 2000 paper is already of record here, a further copy is provided for the Examiner's convenience as Exhibit E. The non-obviousness of the claimed invention and of the underlying work disclosed in the application can thus be further shown by the influence within the scientific community of the paper published by the inventors and their co-workers dealing with the subject matter of this invention; i.e., the Fallon et al. PNAS 2000 article of Exhibit E.

The influence of the Fallon et al. PNAS 2000 article on the inventors' peers and the scientific community is manifested in part by the number of citations made in the published literature to Exhibit E. Footnote 10 found on p. 374 of Exhibit D notes that "[m]uch research has been done showing

that the number of citations a scientist's work receives is a roughly valid indicator of its quality." "Quality", here, translates to unobviousness in that a nicely done but obvious study rarely gets cited by others.

Information as to the number of times that a work is cited in the scientific literature is available in journals and in various databases. The citations to the article of Exhibit E as reported in the Science Citation Index and in the Current Contents series of publications, both of which constitute the Scisearch® database of Knight-Ridder Information Service, formerly the Dialog® Information Retrieval Service, are compiled in the copy of the printout attached as Exhibit F.

As can be seen, there were one-hundred sixty-four citations to the Fallon et al. PNAS 2000 article in the time period after its publication date of December 19, 2000 through the third week of October 2007, with forty-six articles being published in 2001 and 2002. The complete list of citations and abstracts is provided as Exhibit F. All but two of the one-hundred sixty-four publications citing Fallon et al. PNAS 2000 were authored by someone other than an author of Fallon et al. PNAS 2000.

The Coles' article [Exhibit D] points out (page 372) that

"(i) about one-half of all the papers published in the more than 2100 source journals abstracted in SCI [Science Citation Index] do not receive a single citation during the year after it is published; (ii) the average cited author in the 1965 SCI received a mean of 6.08 citations to his life's work."

Of the articles of Exhibit F, twelve were published in 2001 by non-Fallon et al. PNAS 2000 authors. Thus, the work of

applicants has far exceeded both findings (i) and (ii), above, by the Coles article.

As still further evidence of the non-obviousness of the work in the article of Exhibit E as measured by the scientific community, a comparison can be made to the citations to the published work of Nobel Laureates Kohler and Milstein in which their Nobel Prize-winning production of useful monoclonal antibodies was first described. That paper, *Nature*, **256**:495-497 (1975) started the monoclonal antibody industry.

Looking at the period 1975 through 1977, there were a total of thirty-eight citations to the above Kohler and Milstein article. A computer-assisted search similar to that presented for the Fallon et al. PNAS 2000 article in Exhibit F is provided for the Kohler and Milstein article as Exhibit G. Thus, in the comparable time period of two years following the year of publication, there were about the same number of citations to both the inventors' paper (forty-six) and the Kohler and Milstein paper (thirty-eight), with all but two of the citations to the Fallon et al. PNAS 2000 article being cited by 'others' (i.e. non-authors of the Fallon et al. PNAS 2000 article). The number of Kohler and Milstein self-citations is not known.

It is submitted that the above comparison of numbers of citations shows that the work of the Fallon et al. PNAS 2000 paper that is embodied in these claims influenced the authors' scientific peers to an extent comparable to the work of Nobel Laureates Kohler and Milstein. The magnitude of those influences is further submitted to demonstrate that neither work was obvious at the time it was published in that if indeed it were obvious and run-of-the mill, other members of the scientific community would not have deemed it necessary to recognize the articles in their own papers and to base their own work thereon. It is thus submitted that the claimed subject

matter was not obvious to a worker of ordinary skill at the time of the parental filing in 1998, and as such, this basis for rejection should be withdrawn.

Copies of Exhibits C-G are enclosed as part of an IDS, and the exhibits and documents are listed on enclosed Form PTO/SB/08B.

**B. Specific Citations in the peer-reviewed literature  
establishing the significant scientific contribution  
of the methodology  
and results of Fallon et al. PNAS 2000**

In 2001, a report to Congress was prepared by the National Institutes of Health entitled "*Stem Cells: Scientific Progress and Future Research Directions*". The purpose of the report was to provide "a review of the state of the science of stem cell research as of June 17, 2001. Included in this report is subject matter addressing stem cells from adult, fetal tissue, and embryonic sources". The relevant portion of the report referring to the Fallon et al. PNAS 2000 article, Chapter 8, is attached as Exhibit H. For the Examiner's reference, a comprehensive version of the stem cell report is available on the NIH website at <http://stemcells.nih.gov/staticresources/info/scireport/PDFs/fullrptstem.pdf>. The Fallon et al. PNAS 2000 article, cited below as reference [4], is cited in the section of Chapter 8 entitled "*Turning on the Brain's Own Stem Cells as a Repair Mechanism*", a portion of which is included below:

[r]ecent research shows the direction that this may be heading for Parkinson's Disease. James Fallon and colleagues studied the effects on rat brain of a protein called transforming growth factor alpha (TGF $\alpha$ )—a natural peptide found in

the body from the very earliest stages of embryonic development onward that is important in activating normal repair processes in several organs, including liver and skin. Fallon's studies suggest that the brain's normal repair process may never be adequately triggered in a slowly developing degenerative disease like Parkinson's and that providing more TGF $\alpha$  can turn it on. Specifically, Fallon found that TGF $\alpha$  injected into healthy rat brain causes stem cells in the subventricular zone to proliferate for several days, after which they disappear. But if the researchers make similar injections into rats in which they first damage the nigro-striatal neurons with a toxin called 6-hydroxydopamine—a frequently used animal model for Parkinson's Disease—two things happen. After several days of cell proliferation, Fallon observes what he calls a "wave of migration" of the stem cells to the damaged areas, where they differentiate into dopamine neurons. Most importantly, the treated rats do not show the behavioral abnormalities associated with the loss of the neurons. Whether the beneficial effect on symptoms is the result of the newly formed cells or some other trophic effect is not yet entirely clear [4]. [Emphasis supplied, Note: Fallon et al. PNAS 2000 article is reference number 4.]

In a 2006 Research Report published in *Brain Research* 1091:258-264 (attached as Exhibit I), authored by Robert H. Miller entitled "The promise of stem cells for neural repair", the Fallon et al. PNAS 2000 paper is addressed under the section entitled "induction of endogenous neurogenesis for repair" where it states:

[i]nfusion of transforming growth factor-alpha (TGF- $\alpha$ ) in animals with a selective lesion of the dopaminergic nigrostriatal system induces massive proliferation of forebrain stem cells, followed by migration of both glial and neural progenitors toward injection side (Fallon et al. 2000). This treatment resulted in increased numbers of

neurons in the striatum and functional improvement. These findings indicate that a lesion itself is not sufficient to "activate" endogenous precursors and further supports the hypothesis that sensitivity of endogenous neural stem cells to microenvironmental signals occurs during a temporal window provided by pathological conditions and/or by administration of exogenous molecules. [page 261; Emphasis supplied.]

In a 2007 review published in *Nature Reviews Neuroscience*, Ghashghaei et al., 8:141-151 (attached as Exhibit J), entitled "Neuronal migration in the adult brain: are we there yet?" there is a specific citation to the Fallon et al. PNAS 2000 article under the section heading "Migration in ageing, injury and disease" that states:

New neurons or neural precursors can potentially migrate along myelinated fibres, blood vessels and astroglial processes towards the sites of injury using signals such as stromal-cell-derived factor-1 $\alpha$  (SDF1 $\alpha$ ) released from neurons or glia at the sites of injury or by expressing matrix metalloproteinases (for example, MMP9) that can modulate components of the brain ECM in the path of migration<sup>139-145</sup>. [Emphasis supplied; Note: Fallon et al. PNAS 2000 is reference number 139.]

In another 2007 review published in *Gene Therapy and Regulation*, 3(1):91-109 (attached as Exhibit K), authored by Khalid Shah, entitled "Neural Stem Cells and Armed Derivatives: Fate and Therapeutic Potential in the Brain", the teachings of Fallon et al. PNAS 2000 are cited under the section heading 'Cell Repair and Replacement Therapy: Endogenous Repair' wherein that author states:

[i]n one study, the TGF $\beta$  [sic - should be TGF $\alpha$ ] was shown to induce NSC [Neural Stem Cell] proliferation, migration and differentiation in



vivo, as well as behavioural improvement in rats with 6-hydroxydopamine lesions of the *substantia nigra*.

It is thus submitted that the contributions of Fallon et al. PNAS 2000 illuminated in the citations of peer-reviewed articles written by those of at least ordinary skill in the art acknowledge the significant contribution that the discoveries of Fallon et al. PNAS 2000 disclose in regard to the proliferation, differentiation, and most importantly the migration of newly formed neural progenitor cells to a site of injury of existing damage in the central nervous system via administration of purified TGF $\alpha$  polypeptide to a site outside the ventricles.

It is further submitted that the number of citations and unsolicited favorable attention given the work underlying this application illustrate the non-obviousness of the claimed subject matter. The Rejection under 35 USC §103 should therefore be withdrawn.

#### **IV. Related Applications**

In view of the recent ruling from the US Court of Appeals for the Federal Circuit regarding *McKesson Information Solutions, Inc. v. Bridge Medical, Inc.*, 487 F.3d 897 (Fed. Cir. 2007), it is noted that a parental application, U.S. Patent Application Serial No. 09/129,028, of which the present application is a continuation-in-part, is currently pending and on appeal to the USPTO Board of Patent Appeals and Interferences. A second continuation-in-part application, U.S. Patent Application Serial No. 10/167,384, also claiming priority to the '028 application is currently undergoing examination by the USPTO.

V. Summary

Claims 1, 2, 5, 33, and 63 have been amended to speed prosecution and further clarify the claimed subject matter. Further bases for holding the formerly appealed claims to be neither anticipated nor obvious have been provided, as well as an IDS listing the cited art.

It is therefore believed that this application is in condition for allowance of all of the pending claims. An early notice to that effect is earnestly solicited.

No further fee or petition is believed to be necessary. However, should any further fee be needed, please charge our Deposit Account No. 23-0920, and deem this paper to be the required petition.

The Examiner is requested to phone the undersigned should any questions arise that can be dealt with over the phone to expedite this prosecution.

Respectfully submitted,

By

  
Edward P. Gamson, Reg. No. 29,381

Date

January 24, 2008